l'INSERM, Paris, France. Percutaneous kidney biopsies were performed on 30 adult patients with apparently benign proteinuria. Urinary sediment, intravenous pyelography and kidney function were normal in all cases. A mild hypertension followed in 13 cases (mean age: 27 years) the discovery of proteinuria. In 7 patients focal glomerulosclerosis was found on biopsy. In all cases but two there was localised or diffuse arteriolar endarteritis. Arteriolar lesions were the only detectable histologic abnormality in 23 cases of which 14 (mean age: 21 years) had normal blood pressure. Our data and similar ones reported by other authors are in favour of a primary arteriolar renal disease of unknown origin associated with proteinuria.

The Renal Association, London, England, October 25, 1972

The recurrent haematuria syndrome. *R. P. Burden, L. J. Booth, W. N. Boyd, B. G. Ockenden and G. M. Aber, Renal Research Unit, North Staffordshire Hospital Centre.* Clinical features, renal function and histology have been correlated with selective renal angiographic appearances in twenty patients with either recurrent painless haematuria or recurrent loin pain with haematuria, in in whom there was no evidence of urinary infection, systemic disease or a structurally abnormal urinary tract. Renal function has remained normal in all patients for periods of up to five years. Histologically, whereas all patients showed similar glomerular changes consisting of focal and segmental mesangial thickening and proliferation with pericapsular fibrosis, flattening of tubular epithelium was more common in the patients with loin pain and in one case thickening of arteriolar walls was also seen. Renal angiography was normal in those individuals with painless haematuria but all those with loin pain and haematuria showed evidence of focal ischaemia and sometimes cortical infarcts. The differences between the two groups of patients and possible aetiological factors are discussed.

Seasonal nephrotic syndrome. *W. G. Reeves, J. S. Cameron, C. S. Ogg, Guy's Hospital and the Royal Postgraduate Medical School, London.* The association between the relapsing nephrotic syndrome and specific allergy has only been recorded in a few cases (Hardwicke et al, 1959; Wittig and Goldman, 1970) but is of great interest. We know of at least five patients with seasonal exacerbations of their nephrotic syndrome; we have investigated three of these in detail. In each, the nephrotic syndrome was associated with allergy to grass pollen. We concentrate here on detailed findings in one of those patients—a boy aged 11 years when he first had the nephrotic syndrome in 1968. Various parameters were measured regularly from the autumn of 1969 to July 1972, and the seasons 1970 and 1971 have been analysed in detail. The main relapse of the syndrome occurred in June and July of each year, and the seasonal features of note in the plasma were: a fall in total and antigen-specific IgG; a rise in total and antigen-specific IgE; a fall in C3; a preliminary rise in IgA, and a positive C1q test for circulating antibody-antigen complexes. A renal biopsy was performed at the time of the 1971 relapse and showed a "minimal change" pattern on light microscopy, the only feature of note being a rather diffuse increase in thickening of the basement membrane on electron microscopy, most areas examined being 500 to 600 nm. Immunofluorescent staining failed to reveal any IgG, IgM, IgA, IgD and C3, fibrinogen or grass pollen antigen. IgE was sought using two different antisera, and both direct and indirect techniques, but again none was detected. Early in 1972 a course of desensitisation was given. During the following pollen season the patient had mild rhinitis but his urine remains protein-free. Another boy was treated with cyclophosphamide 3 mg/kg for 8 weeks in 1970; although he had only mild proteinuria for the past two grass pollen seasons he still suffers from severe hay fever. The total and antigen-specific IgE still show sharp rises. The third man first suffered a seasonal nephrotic syndrome in 1959, and now has persistent proteinuria, worse in June: his proteinuria also increases after corticosteroids are withdrawn. Possible mechanisms involved in the pathogenesis of this syndrome will be described, and their relevance for atopic diseases and the "minimal change" relapsing nephrotic syndrome discussed.

In vitro synthesis of rat glomerular basement membrane in nephrotoxic glomerulonephritis (NTN). *M. R. Daha, J. de Graeff and A. A. H. Kassenaar, Departments of Nephrology and Chemical Pathology, University Hospital, Leiden, The Netherlands.* The purpose of this study was to investigate the glomerular basement membrane (GBM) synthesis of normal and nephritic rats. Glomerulonephritis was induced by intravenous injection of rabbit-anti-rat-GBM-antiserum. Glomeruli for in vitro studies were isolated in the cold using the method of Krakower and Greenspon as modified by Spiro (1967). As an index of GBM synthesis the proline incorporation in the collagen of the GBM was used. The isolated glomeruli were incubated for seven hours in buffer containing ^{14}C- proline. Glucose oxidation was linear up to 12 hr for normal glomeruli and up to 7 to 8 hr for NTN glomeruli. Two different incubation media were used: medium I, consisting of a Krebs-Ringer phosphate buffer; medium II, a Krebs-Ringer buffer with HEPES but without phosphate. Normal glomeruli thus incubated showed only minor changes by light and electron microscopy. The GBM synthesis, as measured by ^{14}C-proline incorporation was 722 $\pm$ 60 dpm/100 µg DNA for normal glomeruli using medium I. This value was 1750 $\pm$ 92 dpm 24 hr after induction of NTN. In medium II these values were respectively 6671 $\pm$ 309 and 10257 $\pm$ 257. The GBM synthesis, in medium II was maximal between 6 and 16 hr after induction of NTN. A correlation was found between the amount of kidney-fixing-antibodies administered and the maximal stimulation of GBM synthesis. We conclude that after induction of NTN an initial stimulation up to 100% of GBM synthesis occurs. A correlation between the immunological damage to the GBM and GBM synthesis seems to exist.

Immunoglobulin classes and complement components in glomerular deposits. *Professor Ag. Jean Berger. Hopital Necker, Paris.* Complex-type nephritis is a broad concept in which are embraced several forms of glomerulonephritis (GN) which are quite different from a clinicopathological point of view. The composition of the deposits seems to define more precisely each type of GN. Some examples are as follows: 1) Chronic focal GN with haematuria is characterized by diffuse mesangial deposition of IgA, IgG, and C3. 2) The deposits in Henoch-Schonlein purpura nephritis contain IgA, IgG, C3 and fibrinogen. 3) IgM is the main component of the deposits in focal glomerulosclerosis. 4) The humps of acute GN contain IgG and much C3. On the other hand, the deposits in membranous GN contain IgG and little C3. 5) Deposition of C3 without immunoglobulins or early complement components appears to define a special form of membranoproliferative GN. 6) Clq is often prominent in lupus nephritis. It must be stressed that the composition of the deposits remains identical during the whole course of the disease and that the first indication of recurrence in kidney transplants is the deposition of complexes with the same composition as in the patient's own kidneys.

Renal haemodynamic studies and haemodynamic studies in terminal uraemics before and after diuretic therapy. *Ugur Ulku and Kemal Onen, Cerrahpasa Medical Faculty, Istanbul University, Turkey.* The renal excretory patterns of solutes, renal functional and haemodynamic changes were investigated in 9 patients with end-stage kidney failure before and after 200 mg of furosemide i.v. during renal vein catheterisation. In six of the patients cardiac output was also determined. After furosemide, the values for V, $U_{Na}V$, U_KV, $U_{Cl}V$ and GFR (8.68 ml/min) increased significantly. A slight rise of C_{H_2O} (from 0.1 to 0.35 ml/min) was observed and may be explained by the increased solute load to the counter-current multiplier and by a slight increase in its already depressed activity. The high values for C_{Na}/GFR ratios (0.02) values suggest that the glomerulotubular balance is significantly altered and that the tubular fractional absorption of sodium is depressed. The high C_{Na} and cardiac output values (mean 7.6 L/min) suggest that the volume dependent mechanism for natriuresis is active. C_{PAH} (52.3 ml/min) and RPF (184 ml/min) as calculated by the determination of E_{PAH} (C_{PAH}/E_{PAH}) were different and Tm_{PAH} were found to be 5.88 and 11.8 mg/100 ml before and after furosemide respectively. The filtration fraction (FF) was extremely low (0.045). This suggests very low oncotic pressure in post glomerular peritubular capillaries which might be a factor working against the reabsorption of sodium. Arterial and renal venous O_2 differences were 2.1 and 1.7 before and after furosemide (normal 1.5−1.7). Total O_2 uptake by the kidney before and after furosemide was 5.02 and 4.05 ml/min. The differences were not statistically significant. T_{Na}/V_{O_2} values were found 19.1 and 22.4 before and after furosemide. This suggests that the effect of furosemide is on the membrane transport of Na.

The renal clearance of a new amino acid, homoarginine, in normal subjects and patients with cystinuria. *B. D. Cox, D. Calame and J. S. Cameron, Guy's Hospital, London, England.* Cystinuria is characterised by the hyperexcretion of four dibasic amino-acids: cystine, ornithine, arginine and lysine. Frimpter (1965) showed that in addition to these four aminoacids, patients with cystinuria also excreted the mixed disulphide of cystine and homocystine. Ion exchange chromatography of urinary guanidine compounds and aminoacids in 7 stone-forming cystinuric patients showed that all were excreting an unidentified Sakaguchi-positive amino acid in addition to the five compounds just discussed. Comparison with standard compounds suggested that this was homoarginine, previously reported in leguminous plants. Examination of urine from five normal subjects showed that homoarginine is also excreted in health, but in much smaller quantities. After concentration, plasmas from both normal subjects and the cystinuric patients were shown to contain homoarginine in quantities of about one hundredth that of arginine, the levels being somewhat lower in the cystinuric patients' plasma. The renal clearance of homoarginine was higher than that of arginine in normal subjects, but although the clearance was many times greater for both compounds in the cystinuric patients, in all those studied the clearance of arginine exceeded that of homoarginine. These findings support the suggestion of Dent and Rose (1951) that the tubular reabsorptive site (or sites) which are defective in cystinuria require an α-amino acid group at one end of the molecule and another amino group at the other with a chain of 4–6 carbon atoms between; and that yet other compounds which satisfy these requirements might be excreted in excess in cystinuric patients.

The effect of caloric intake on nitrogen balance in chronic renal failure. *B. E. B. Hyne, Edna Fowell, and H. A. Lee.* Nitrogen balances were carried out on 7 adult patients in stable chronic renal failure. Diets were designed to give patients a nitrogen intake of between 40 and 54 mg/kg body weight. High biological value protein accounted for more than 70% of the protein allowance. The caloric intake was kept constant at any one study and ranged between 36 and 55 calories/kg body wt. Carbohydrate provided between 50 and 70% of the total caloric intake. No correlation was found between net nitrogen intake and nitrogen balance over the range studied. A highly significant correlation was obtained between caloric intake and nitrogen balance. Nitrogen balance in uraemic patients on similar nitrogen intakes improves with increasing caloric intake in the range 36–55 calories/kg body wt. Patients in advanced chronic renal failure, unlike normal individuals, show improvement in nitrogen balance on low nitrogen intakes (3–4 g/day) when the caloric intake is increased above 25 calories/kg body wt. The degree of the improvement in nitrogen balance is compatible with increased utilization of endogenous nitrogen probably as a result of increased dietary calories.

Plasma renin, exchangeable sodium and vascular reactivity in patients with terminal renal failure. *S. M. Rosen and P. J. A. Robinson, Department of Renal Medicine, Leeds University Hospital and the General Infirmary at Leeds, Leeds, England.* Plasma renin and exchangeable sodium were measured in two groups of patients with terminal renal failure treated by maintenance dialysis therapy. Group A consisted of seven patients with a mean blood pressure less than 110 mm Hg, and group B consisted of five hypertensive patients whose mean blood pessure was higher than 116 mm Hg. Mean plasma renin was higher in the hypertensive group. The higher levels of plasma renin in group B could not be the sole reason for the hypertension, since plasma renin is known to rise during dialysis in spite of the decrease in blood pressure. Although blood pressure could be reduced by the reduction of sodium in individuals during dialysis, the mean value for exchangeable sodium was marginally lower in the hypertensive group. In group A, there was an inverse correlation between plasma renin and exchangeable sodium, suggesting that the 'end-stage' kidney still responds to removal of sodium by release of renin. This correlation did not exist in group B, in which there was a higher concentration of plasma renin for a given level of exchangeable sodium. Vascular reactivity of forearm blood vessels in these patients was measured by infusion of noradrenaline into the brachial artery and determining the threshold dose required to produce an alteration in blood flow in the ipsilateral forearm. The threshold dose increased as the level of renin decreased for a given value of exchangeable sodium. These results support the hypothesis that the effect of renin on blood pressure is dependent on levels of exchangeable sodium and hypertension ensues when there is an inappropriately high concentration of plasma renin.

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Role of the kidneys in the inactivation of circulating renin. *L. Peters-Haefeli and G. Peters, Institut de Pharmacologie de L'Universite de Lausanne, Switzerland.* Previous experiments in this laboratory showed that nephrectomy lowers the total clearance of rat renin in rats from 2.9 ml/kg/min to 1.6 ml/kg/min. The kidneys thus appear to be responsible for 44% of the total amount of renin inactivated. While the total clearance of heterologous (hog) renin in the nephrectomized rat was the same as that of rat renin, the total clearance of the heterologous renin in intact rats was much smaller (2.0 ml/kg/min). Rat kidneys thus